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COMPARATIVE STUDY OF INTRAVENOUS DEXMEDETOMIDINE AND INTRAVENOUS MAGNESIUM SULPHATE TO ATTENUATE HEMODYNAMIC STRESS RESPONSE TO LARYNGOSCOPY AND INTUBATION

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INTRODUCTION

The term intubation has been first described over a century ago by Rowbatham and Magill is still being in-practice. In general, various hemodynamic changes are quite common followed by induction of general anaesthesia, laryngoscopy, tracheal intubation, and extubation. In the 1940s, Reid and Brace had first described the hemodynamic responses followed by laryngoscopy and intubation [1]. Subsequent to the above study, King et al., in the year 1951 have also well documented the same. Laryngoscopy and tracheal intubation are most commonly associated with sympathetic stimulation which leads to tachycardia and hypertension which might eventually predispose to myocardial ischemia [3]. It is quite common that hypertensive response to anesthetic induction with endotracheal intubation would be harmful to patients those who have been diagnosed with cardiovascular disease, increased intracranial pressure, or anomalies of the cerebral vessels.

In general anaesthesia, laryngoscopy and tracheal intubation are the most essential parts, but severely noxious stimuli induce an intense sympathetic hemodynamic response [4]. Normally an imbalance in myocardial oxygen supply and demand, predisposing patients to ischemia, infarction and heart failure are caused by tachycardia and hypertension [1]. Such response to laryngoscopy and endotracheal intubation are due to intense sympathetic discharge that are caused due to stimulation for the upper respiratory tract, both during laryngoscopy and endotracheal intubation which normally gives rise to changes in the concentrations of plasma catecholamines [5]. Though these sympathetic responses are normally being tolerated by healthy patients, it can be quite hazardous in patients with compromised cardiovascular function. In addition, multiple investigations have revealed that the afferent signal travels to the brain stem via the glossopharyngeal and vagus nerves, generating autonomic nervous system activation and tachycardia and hypertension. Following intubation in hypertensive patients, increased rates of cardiac arrhythmias, myocardial ischemia, abrupt left ventricular failure, and cerebrovascular accidents are more likely as a result of the aforesaid problems [6][7]. Some of the most common factors that might precipitate the laryngoscopy and intubation mediated pressor response include light planes of anaesthesia, prolongation in the time of procedure, elevation of vagally innervated posterior part of the epiglottis by straight/Miller blade, difficult view, force used to displace the tongue and further manipulations during laryngoscopy and intubation [8]. Hence, it is an essential

part of anaesthesia to consider pre-operative sedation and cardiovascular stability during induction, laryngoscopy, intubation and further throughout the surgical procedure for a better patient outcome.

Therefore, recommendations for attenuation of reflex hypertension and tachycardia by upper airway irritation has been a great challenge. Despite decreasing the cardiovascular response, patients at risk must also satisfy some of the basic requirements before even anaesthesia induction. Those includes the applicability regardless of patient collaboration, prevention of impairment of cerebral flow, and avoid arousal of the patient and further it should neither be time consuming nor affect the duration of subsequent anaesthesia [9]. Even though several methods have been identified to attenuate these hemodynamic response to laryngoscopy and intubation since the date of its recognition, still suitable methods to attenuate the response is under search.

Since the middle of 20th century attempts have been made by Reide & Brace and other investigators to reduce the laryngoscopy and intubation mediated sympathetic response, which includes,

- Deepening the plane of anaesthesia with the use of agents such as inhalational and intravenous anaesthetics [2].
- Reducing the total laryngoscopy duration to less than 15 seconds.
- Use of synthetic drugs such as opioids, lignocaine, vasoactive drugs like calcium channel blockers, nitroprusside and other drugs primarily clonidine and dexmedetomidine called as alpha 2 agonists.[10][11].

Even though numerous pharmacological techniques have been assessed to attenuate the serious adverse hemodynamic responses to laryngoscopy and tracheal intubation, including deepening the anaesthesia and, pre-treatment with opioids, (beta) blockers, vasodilators and calcium channel blockers no concrete results have been achieved still [4]. Non-opioid medications have recently received increased attention as a part of a multimodal regimen for reducing anxiety and intubation reaction. Its usefulness in decreasing the dose of parenteral analgesics and providing post-operative pain relief has been thoroughly proven in various studies [12][13][14]. Pre-treatment using beta blockers, calcium channel blockers, nitroglycerine, opioids, gabapentin, dexmedetomidine, and clonidine instead of cholinergic premedication is a widespread technique with mixed effects [15][16][17][18][19].

In the recent past, pregabalin and gabapentin, the gamma-amino butyric acid (GABA) analogues, have gained the in sight of prominence in various clinical studies as they not only alleviate perioperative pain but also effectively compromise the pressor response caused due to intubation and further produce perioperative sedation and anxiolysis. In the year 1993 gabapentin was introduced as antiepileptic drug. It is also reported that gabapentin has the effect of attenuating the hemodynamic response to laryngoscopy and intubation [20].Yet another GABA analogue called the pregabalin is a lipophilic compound with good absorption rate and well tolerated after oral administration with very limited side effects that doesn't have any known clinically relevant drug interactions. The hepatic metabolism of pregabalin is insignificant and normally eliminated by renal excretion. Additionally, it is said to have anxiolytic, analgesic, and anticonvulsant effects. Drugs like gabapentin and pregabalin have been shown in recent studies to reduce stress caused by laryngoscopy and intubation. Several studies have established the efficacy of oral pregabalin for postoperative analgesia and the decrease of parenteral analgesics [21][22]. However, only a few studies on the cardiovascular effects of pregabalin on patients following surgery have been published and are available in the literature Rastogi Bhawan et al., 2012; Ebru Salman et al., 2012. However, the drugs and manoeuvers for mitigating the stress response used have variable benefits and side effects [25]

Search for an ideal drug that can attenuate the pressor response to intubation is being one of the emerging need. Hence, there is a need for identification of not only an appropriate premedication but also for smooth induction and rapid intubation that would prevent any associated risks and complications of the hemodynamic pressor responses to weaken it pharmacologically.

Dexmedetomidine, a central α-2 agonist is one of the premedicant used as intravenous anaesthesia. The advantages of using Dexmedetomidine as premedicant during anaesthesia procedures includes sedation, analgesia, anxiolysis and improve hemodynamic stability. Due to afore said benefits of Dexmedetomidine, minimum alveolar concentration have been identified and therefore the required amount of anaesthetics would also be decreased [11]. Previous study has also reported that it could minimize the hemodynamic response to laryngoscopy and intubation [26][27].

Another commonly used pressor attenuating premedicant for anaesthesia is magnesium sulfate ($MgSO_4$), which blocks the release of catecholamines from the adrenergic nerve terminals, increased magnesium sulfate could inhibit the release of catecholamines. It also directly acts on

the blood vessels and causes vasodilation and thereby attenuates vasopressin-mediated vasoconstriction.

Even though in various other countries Dexmedetomidine is being used as premedicant since years, in India it has been introduced in the year 2009 only. However, based on literature search and up to our understating, study reports on using Dexmedetomidine and magnesium sulfate in our country is seldom available.

This study was done to compare the effectiveness of intravenous Magnesium sulphate and Dexmedetomidine for attenuation of pressor response during laryngoscopy and endotracheal intubation.

Fig.1 Miller's laryngoscope with different blade sizes

Fig.2 Machintosh laryngoscope with different blade sizes

Fig.3 McCoy laryngoscope with different blade sizes

AIMS AND OBJECTIVES

AIM: To compare the effectiveness of intravenous Magnesium sulphate and intravenous Dexmeditomidine in attenuating hemodynamic stress response during laryngoscopy and endotracheal intubation.

OBJECTIVES:

- To compare the effectiveness of intravenous Magnesium sulphate and intravenous Dexmeditomidine in attenuating tachycardia during laryngoscopy and endotracheal intubation.
- To compare the effectiveness of intravenous Magnesium sulphate and intravenous Dexmeditomidine in attenuating blood pressure changes during laryngoscopy and endotracheal intubation.
- To evaluate side effects of intravenous Magnesium sulphate and intravenous Dexmeditomidine.

NEED FOR STUDY:

- Endotracheal intubation is a frequently performed procedure in practice of anaesthesia. It includes direct laryngoscopy and intubation, this produces vasopressor stress response, characterized by tachycardia and increased blood pressure. Till date hemodynamic stress during laryngoscopy and intubation has not been alleviated completely.
- Though Dexmeditomidine has been found to be quite effective in achieving the hemodynamic stability during laryngoscopy and intubation, it is too costly for the majority Indian population, thus there is a need to find a cheaper but equally or more effective drug to achieve a similar effect.

SAMPLE SIZE CALCULATION

Sample size calculation:

$n=2(Z\alpha+Z1-\beta)2\sigma2/(N1-N2)2$

$Z\alpha$= 1.96 at 5% level of significance

$Z1-\beta$= 1.28 at 80% power of study

N1= mean of group D (dexmeditomidine)

N2= mean of group M (MgSO41)

σ= pooled standard deviation

Based on an earlier report (Mona Panchal and Manisha Kapdi, 2018), and using the above given sample size calculation formulae, the required sample size was derived as n=34 in each groups.

n=34 in each group

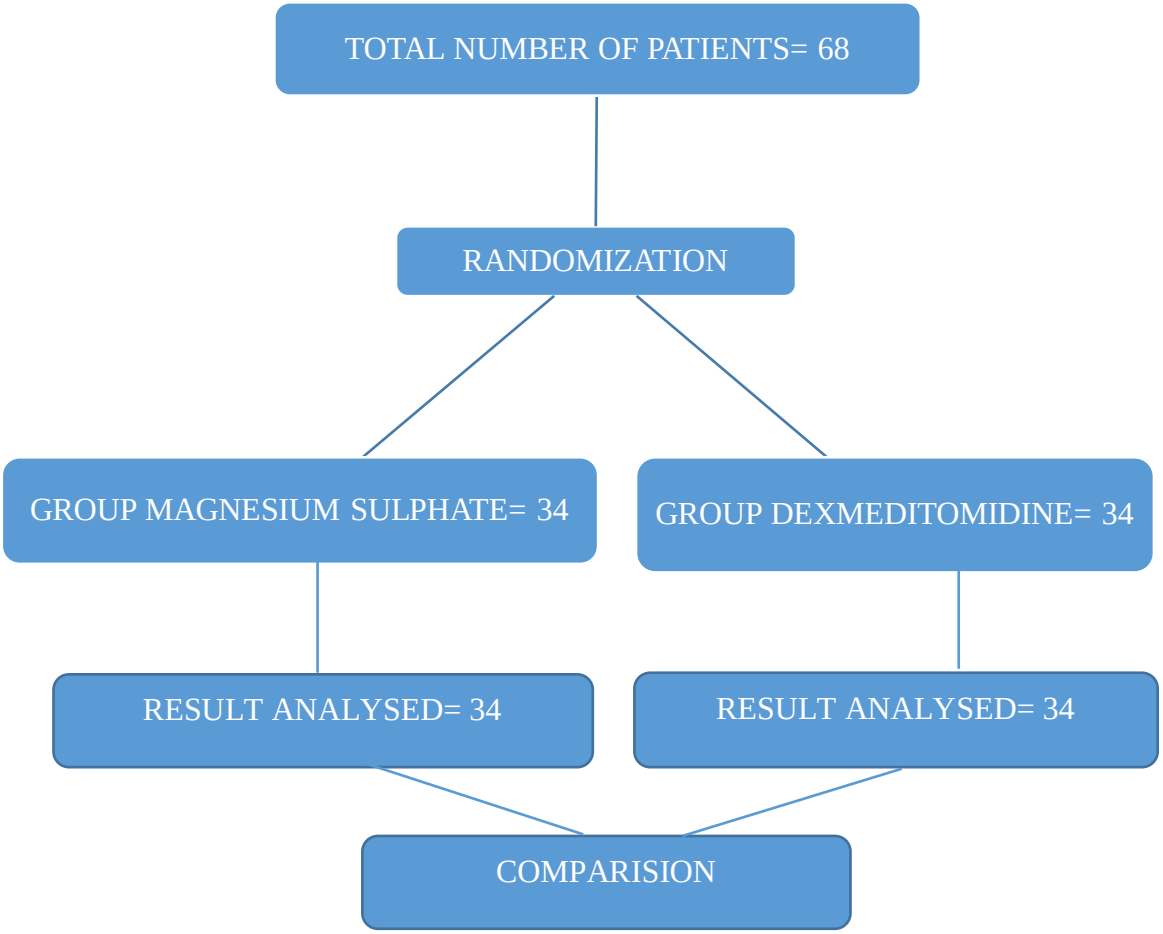
SD= 4.9

Effect size= 3.34

Considering 10% drop out

Total= 68 is the sample size

FLOW CHART OF STUDY DESIGN



REVIWE OF LITERATURE

Laryngoscopy and tracheal intubation are considered to be a powerful noxious stimuli to cause adverse hemodynamic responses such as stimulating undesirable responses in the cardiovascular, respiratory and other physiological manifestations (Henderson, 2010). Responses to airway instrumentation during general anesthesia often results in hemodynamic pressor that leads to hazardous complications eventually. It can lead to clinical manifestation such as tachycardia and hypertension through provocation of a marked sympathetic response. Studies have reported that such manifestation caused due to airway instrumentation could be well tolerated by normal patients but in hypertensive patients it might lead to disastrous events [1][28]. In support of the above cited studies, several other studies have also reported that intubation in hypertensive patients would results in high incidence of cardiac arrhythmia, myocardial ischemia, acute left ventricular failure and cerebrovascular accidents [6][7]. Laryngoscopy and tracheal intubation are thus recognised as noxious stimuli that result in a rise in the autonomic response that lasts just a short time.

In healthy people, it is generally considered to be unimportant and to be followed by a rise in heart rate and blood pressure, but it can be harmful in fragile individuals. The intensity of anaesthesia, the length and complexity of the laryngoscopy and intubation, as well as additional patient features like diabetes and cardiovascular disease, all influence how this reaction develops.

However, pressor response due to intubation is a part of a huge spectrum of stress response that results from the increase in sympathetic and sympatho-adrenal activity that causes a raise in the plasma catecholamines in patients undergoing surgery under general anaesthesia [29]. Normally these changes would be maximum at one minute after intubation and persists for 5 to 10 minutes. The pharyngeal and laryngeal tissue stretching are considered to be the primary cause of these haemodynamic response.

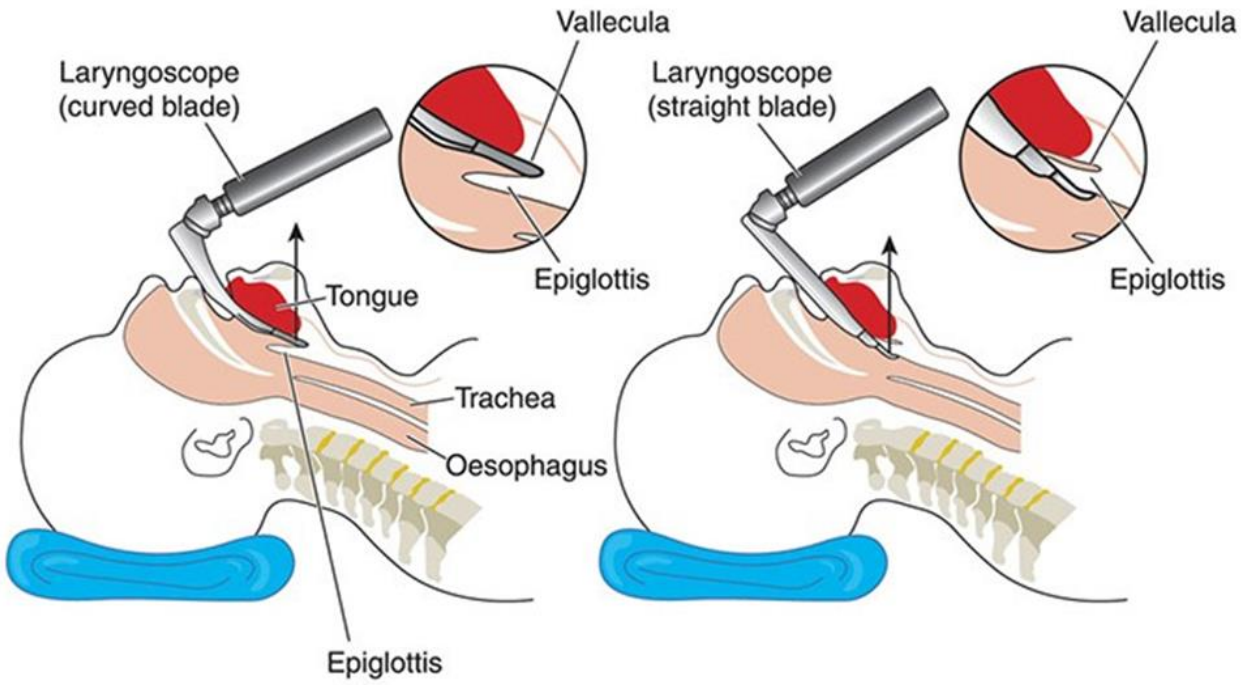


Fig.4 Laryngoscopy

Types of Intubation Responses

Pharynx: Sensory Innervations: The glossopharyngeal nerve supplies visceral sensory fibres to all parts of the pharynx, the anterior epiglottis, the fauces and tonsillae, the posterior 1/3 of the tongue, and the tonsils.

Motor Innervations: The pharyngeal branch of the vagus nerve supplies efferent feed to the pharynx. [30].

The Larynx: Sensory Innervations: The internal laryngeal nerve, a branch of the superior laryngeal nerve, transmits sensory information from the posterior epiglottis to the vocal cords. The recurrent laryngeal nerve supplies both the trachea and the larynx below the vocal cords.

Motor Innervations: The recurrent laryngeal nerve supplies all intrinsic laryngeal muscles, excluding the cricothyroid muscles [30]. Glossopharyngeal sensory innervations supply the oropharynx, the back of the tongue, and the front region of the epiglottis. The distal airway tissues and the posterior epiglottis are supplied by the vagus nerve branches.

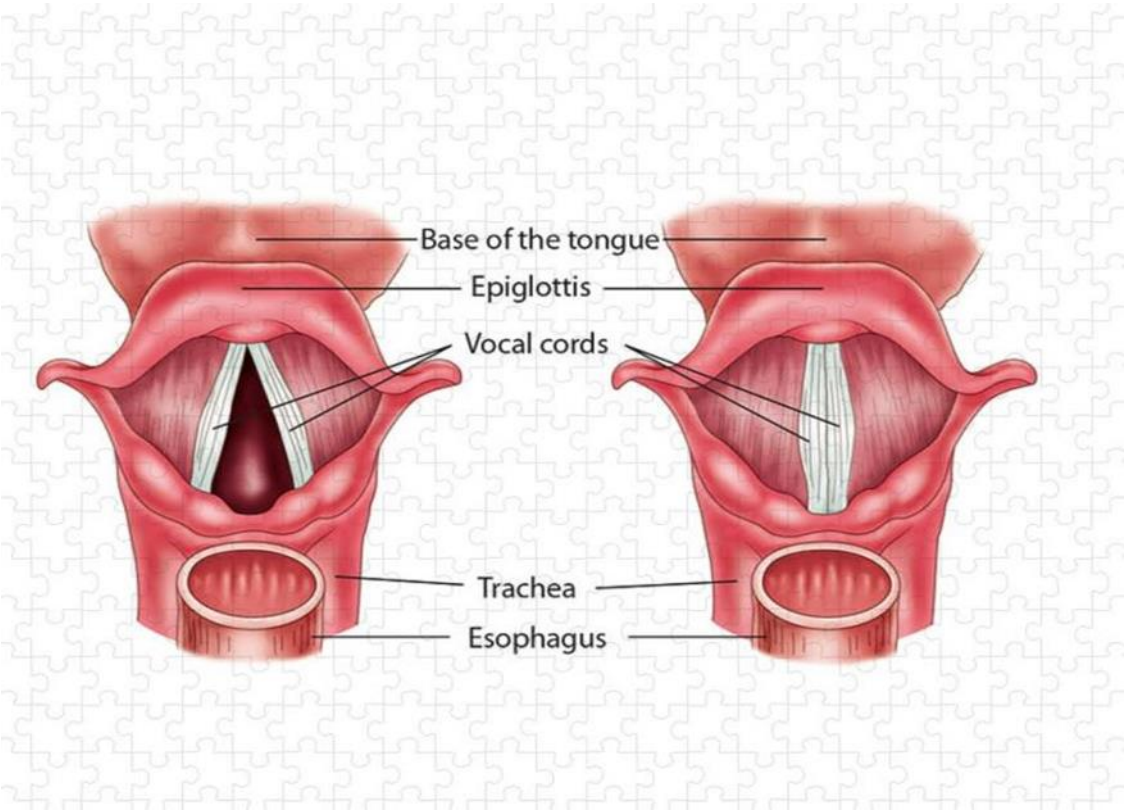


Fig.5 Larynx

Mechanisms involved in Intubation Responses

Intubation reaction (IR) has both sympathetic and parasympathetic components, however, the exact mechanism is yet to be cleared. The consequence of a transitory lasting fewer than 10 minutes following intubation [31].

Sympathetic: The glossopharyngeal and vagus nerves make up the afferent arc that travels from the brain stem and spinal cord to the sympathetic nervous system. An extensive autonomic response is produced at the efferent side due to an increase in the fringe of cardio-accelerator fibres and the production of adrenergic mediators such norepinephrine, epinephrine, and vasopressin.

Parasympathetic: Monosynaptic responses like the parasympathetic reflex are more common in children but can also happen in adults. The response is mediated by increased vagal tone at the SA node [5]. The majority of people perceive the hemodynamic response to laryngeal and endotracheal intubation (LETI) to be transient and negligible. Myocardial ischemia, infarction, arrhythmias, cardiac failure, pulmonary edoema, and cerebral haemorrhage may be linked in patients with coronary artery disease (CAD) to hypertension, increased intracranial or intraocular pressure, and myocardial ischemia, infarction, arrhythmias, cardiac failure, pulmonary edoema, and cerebral haemorrhage [32].

The intubation procedure contains several steps, each of which has a particular effect on the hemodynamic response. There are two stages to orotracheal intubation: Laryngoscopy with direct insertion of an endotracheal tube via the voice chords and into the trachea [33]. Various anaesthetics, adjuvants, and analgesics, on the other hand, have been utilized to reduce the degree of stimulation and the stress reaction to airway manipulation and stimulation during laryngoscopy and intubation.

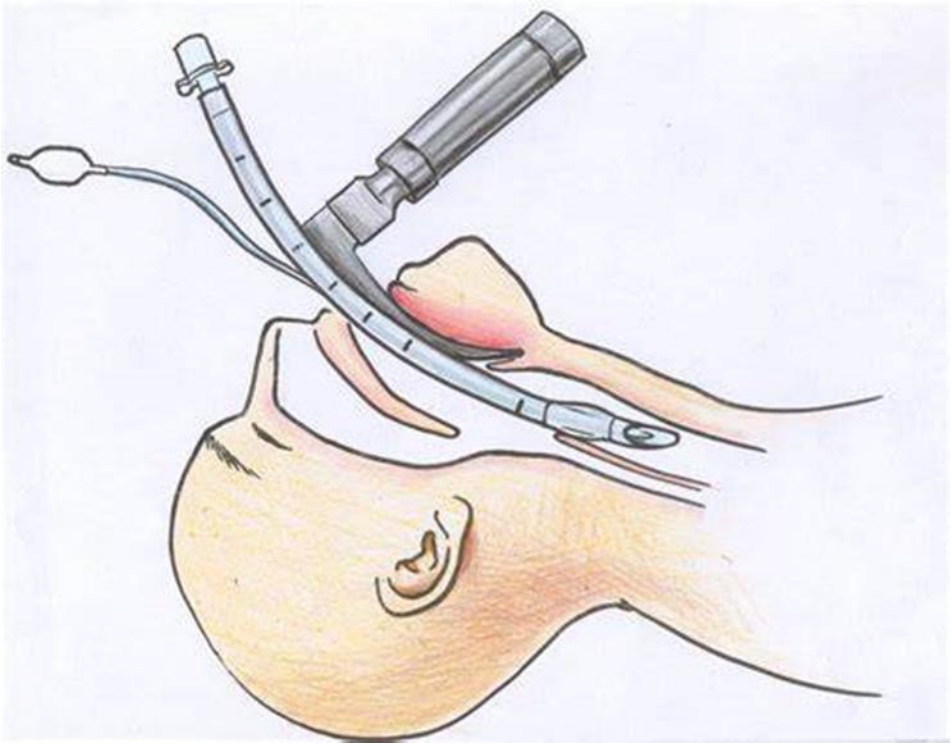


Fig.6 Intubation

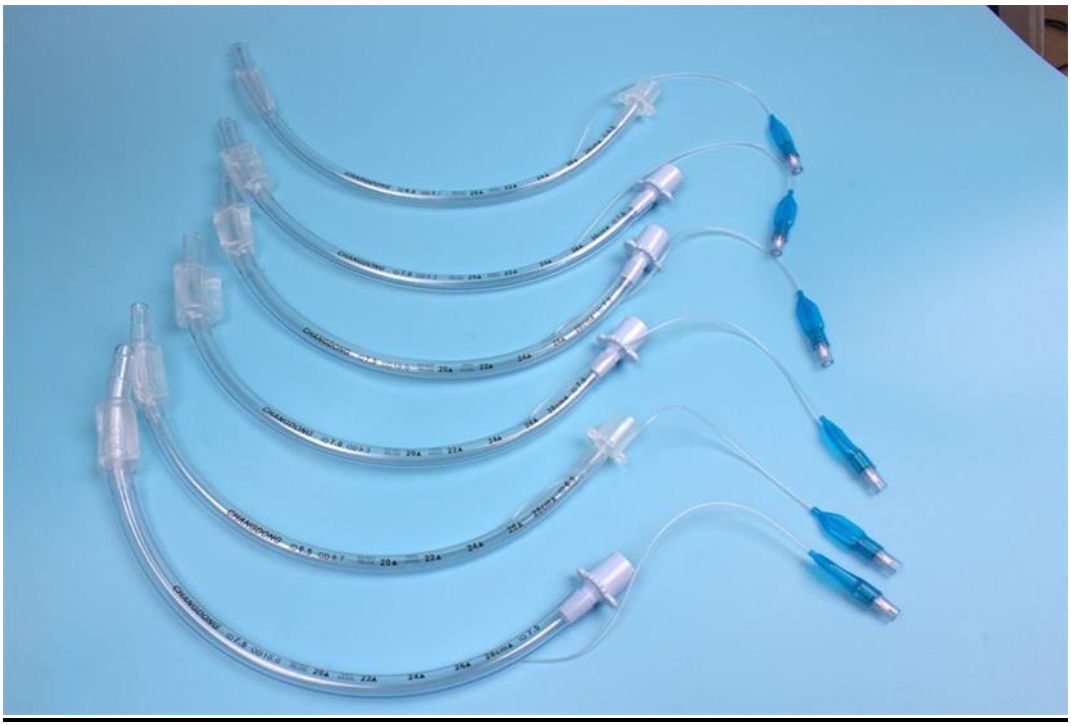


Fig.7 Different sizes of cuffed endotracheal tube

Reducing the Hemodynamic Response

Pharmacological, peripheral blocks, and modifications in method, such as the use of various blades and conduits for intubations, are just a few of the options available to counteract or reduce the effects of intubation. Earlier studies have stated that to blunt these pressor responses, several methods such as deep anaesthesia, topical anaesthesia, opioids, calcium channel blockers, β-blockers, laryngeal mask airway (LMA) have been tried and achieved varying level of success [10][34][35][36][37].

Pharmacological Options:

- Local Anaesthetics
- Opioids
- B2 Blockers
- $MgSO_4$
- α2 Agonists
- Gabapentin, Pregabalin

Lignocaine: It is debatable if lignocaine can effectively reduce the intubation reaction (IR). There are several examples of Lignocaine administration using various dosages, timings, and routes that date back to the 1960s [38].

Opioids: The depth of anaesthesia is increased by adding opioids, which also reduces the sympathetic outflow [39][40].

Alfentanil: For hypertensive patients receiving long-term therapy, it has been reported that a dosage of alfentanil of 10 g/kg body weight administered over 30 seconds can effectively attenuate the cardiovascular impact. [41].

Remifentanil: Remifentanil is an appealing medication for reducing the IR because of its quick onset and brief duration. According to a research conducted by Cha et al. in 2006, a dose of 0.5 g/kg is the ideal dosage for reducing pressor responses connected to the cardiovascular system during endotracheal intubation [42].

Fentanyl: 5–6 g/kg of dosage administered 3–5 minutes before to LETI, with a lower dose for older patients. With these dosages, there is a chance for problems including respiratory depression and, in rare cases, delayed waking [40].

Sulfentanil: Sulfentanil has been studied extensively in the obstetric population, but less is known about its usage in the adult population. For adults, a dosage of 0.5 to 1 g/kg administered two minutes before to LETI was adequate to prevent the reaction [39].

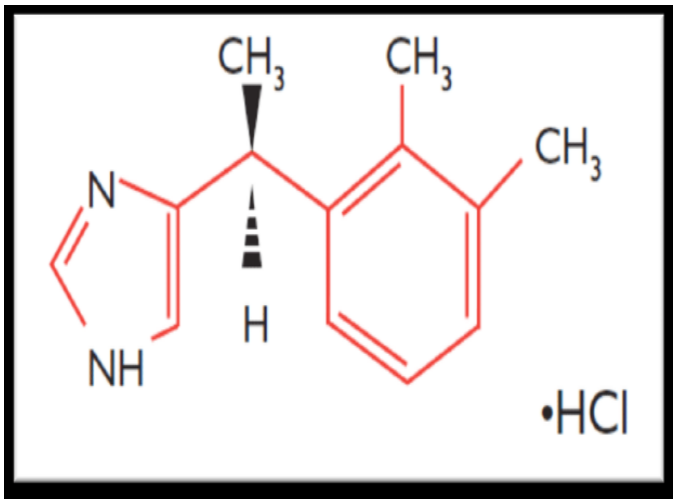
β-blockers: The combination of and blockage is present in labetolol. The blockade is non-specific for blockade and particular for blockade 1. The ratio of a blocking effect to one is one to seven. It has a half-life of up to 5.5 hours and an onset of 1 to 5 minutes. One of the biggest issues with labetalol is its protracted duration of effect. Esmolol is a fast-acting, cardio-selective blocker with a half-life of approximately 9 minutes. It reaches peak blood levels in about 2 minutes. The usefulness of Esmolol in reducing the reaction of LETI has also been examined in a number of trials. Compared to other medications, little to very little agreement has been obtained about the best dosage, timing, and side-effect risk in the various study population groups [38].

Pregabalin: Pregabalin, a β-isobutyl-γ-aminobutyric acid (beta-isobutyl-GABA) and Lyrica, is a GABA genic depressant which, in terms of its pharmacological properties, falls within the GABA pentinoid class of compounds . It is a common prescription drug which is typically used to treat neuropathic pain, anxiety, restless leg syndrome, and as an adjunct drug in the treatment of seizures [43]

Adrenergic α2-agonists: **Clonidine** (2 Agonist) used in addition to a common general anaesthetic. A per oral dosage of 4-5 g/kg given between 60 and 90 minutes before surgery may be helpful, resulting in a decrease in the dose of anaesthesia and sedatives, a reduction in the need for induction drugs, a reduction in the hemodynamic response to LETI, and an improvement in intraoperative hemodynamic. Nor-adrenaline and rennin secretion are decreased by stimulation of the presynaptic receptors. There are also vagotonic effects that cause the HR to drop.

Dexmedtomidine: A highly selective adrenergic 2-agonist that causes a biphasic reaction in the cardiovascular system. It is common to see a brief increase in blood pressure and a drop in heart rate after a bolus of 1 g/kg, which is assumed to be caused by the activation of 2 receptors in vascular smooth muscle. Both the -2BAR and -2AAR produce hypertension and hypotension, respectively [44] As a result, the vasoconstrictive impact manifests prior to the central effects. By administering the bolus dosage as an infusion over 10 minutes, this effect can be lessened but not entirely eliminated [45].

Chemical Structure:



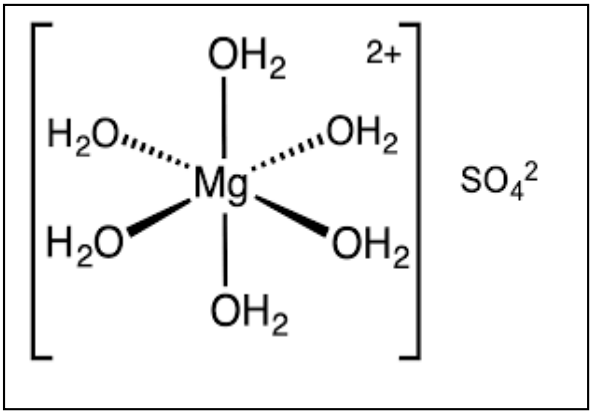
Pharmacodynamics: Dexmedetomidine, an adrenergic α2-agonist, mainly activates 2-adrenoreceptors and thereby causing a decrease in sympathetic tone, which attenuates the neuroendocrine and hemodynamic responses of the patients to anaesthesia and surgery. More interestingly, it causes sedation and analgesia and reduces the requirement of anaesthetic and opioids.

Mechanism of Action: Dexmedetomidine, a specific and selective adrenoreceptor α2-agonist, binds to the presynaptic α2-adernorecptors and thereby inhibits the release of norepinephrine and terminates the propagation of pain signals. Furthermore, with the activation of the postsynaptic α2-adrenoreceptors, Dexmedetomidine could also inhibit the sympathetic activity and decrease the blood pressure and heart rate.

Metabolism and Route of Elimination: Liver is the primary site of metabolism for the Dexmedetomidine via., glucuronidation pathway. Major route of elimination of the administered Dexmedetomidine is identified as almost 95% in through the urine and 4 % through faeces.

Magnesium Sulphate ($MgSO_4$): Control of vasomotor tone, cardiac excitability, and neurotransmitter release are all examples of physiological processes. It induces vasodilation and lowers catecholamine release from nerve terminals and adrenal glands through a variety of mechanisms [46].

Chemical Structure:



Pharmacodynamics: Magnesium Sulphate, a small colorless crystal is being used as anticonvulsant, a cathartic, and an electrolyte replenisher while treating pre-eclampsia and eclampsia. Magnesium Sulphate has the potency of direct inhibitory action in myomaterial muscle cells.

Mechanism of Action: Magnesium, considered to be the second most plentiful cation in the body, is one of the essential cation for the activity of many enzyme systems that plays vital role with regards to neurochemical transmission and muscular excitability. It has the capacity to reduce the striated muscle contractions and further blocks peripheral neuromuscular transmission by decreasing the acetylcholine release at the myoneural junction. In addition all the above, Magnesium inhibits the Calcium influx through the dihydropyridine-sensitive, voltage-dependent channels, which accounts for its relaxant action on vascular muscle.

Metabolism and Route of Elimination: Being an inorganic cation, there is no specific metabolic site for Magnesium. The major route of elimination is solely by kidney through glomerular filtration.

Review of Clinical Studies

The hemodynamic response to laryngoscopy and tracheal intubation, its physiological foundation, the relevance of reducing these circulatory alterations, and the many pharmaceutical treatments for reducing this reaction have all been thoroughly investigated.

Reid and Brace proposed that irritant receptors exist in the larynx, trachea, bronchi, and lungs in 1940. Because both the afferent and efferent fibres are considered vagal, mechanical activation of these receptors causes a vasovagal response, resulting in bradycardia.

In order to determine the function of magnesium in reducing stress reactions brought on by laryngoscopy and intubation, Montazeri and his colleagues recruited 100 ASA-1 individuals between the ages of 15 and 50 for a clinical research. The authors have reported that administration of 30mg kg^{-1} of Magnesium sulphate at the time of intubation of anaesthesia could improve the hemodynamic pressor responses to intubation with les side effects.

In the year 2012, Sulaiman S et al., has enrolled 60 ASA I and II patients between 18 and 60 years those who were scheduled for off pump coronary artery bypass surgery under general anaesthesia and conducted a study to investigate if single dose of Dexmedetomidine could attenuate the stress response to intubation. Patients have been grouped into two, group 1 received 0.5 mcg kg^{-1} of Dexmedetomidine with normal saline and another group of 30 patients received same amount of normal saline. From the observations the authors have concluded that single dose of Dexmedetomidine could effectively decreased the stress response.

A randomized study where 50 patients were divided into two groups conducted by Yildiz et al., to show the effect of Dexmedetomidine at a dose of 1mcg kg^{-1} on stress responses during intubation in comparison with control group. The observations based on Ramsay sedation score scale, vital parameters, adverse drug reactions, patients received Dexmedetomidine showed decreased blood pressure and heart rate.

A study designed and carried out by Salman et al. 2022, to determine the efficacy of Magnesium sulphate on preoperative administration in reducing intubation stress response involved 40 patients randomly categorized into two groups namely Magnesium sulphate 30mg/kg administered group and the Control group where same volume of normal saline was administered. The authors have concluded the stress response due to laryngoscopy and intubation was attenuated with the use of Magnesium sulphate.

Saraf et al., in the year 2013 have investigated if Dexmedetomidine an ideal drug to attenuate pressor response by enrolling 100 patients between the ages of 14 and 55 years old and randomly assigned into two namely, Group C received 10 ml of normal saline and Group D received 0.6µg kg^{-1} Dexmedetomidine dissolved in 10 ml of normal saline. The observations of the study showed

that intravenous injection of 0. 6µg kg^{-1} Dexmedetomidine 10 minutes before intubation was able to effectively blunt the intubation mediated stress response.

Arpita Laha and co-workers have evaluated the attenuation of sympathoadrenal response of patients received 1 µg kg^{-1} of Dexmedetomidine on comparison of their observations with patients received only normal saline. The authors have concluded that administration of 1 µg kg^{-1} of Dexmedetomidine before operation resulted in sedation and blunting of stress response due to intubation.

Gautham Pillai et al., enrolled 80 patients came for spine surgery and grouped into two namely Group M received 30mg kg^{-1} of Magnesium sulphate a bolus along with 10mg kg^{-1} hr^{-1} intravenous continuous infusion and Group C received normal saline. The authors have reported that preoperative intravenous Magnesium Sulphate was able to effectively attenuate the stress response due to intubation when compared with the control group.

A randomized controlled study was conducted by Rajdip Hazar et al., with regard to the hemodynamic pressor attenuation capability of Dexmedetomidine due to intubation in cardiac surgery by selecting 90 ASA II and III patients of age 18 to 45 of both sexes scheduled for elective cardiac surgery. Patients were randomly divided into two namely, Group C called as control received 20ml of saline and Group D received Dexmedetomidine at a dose of 0.5µg kg^{-1} in saline. The authors have confirmed that administration of Dexmedetomidine before anaesthetic induction could blunt the stress response due to intubation.

A study to compare the effect of intravenous 50% Magnesium sulphate and Dexmedetomidine to attenuate the hemodynamic pressor response was designed and conducted by Krishna Chaitanya et al in the year 2014. The authors reported that both Magnesium sulphate at a dose of 30 mg kg^{-1} and 1 mcg kg^{-1} of Dexmedetomidine were able to effectively attenuate the rise in systolic, diastolic and mean blood pressure. Whereas, Magnesium sulphate was unable to reduce the rise in heart rate. However, the authors came to the conclusion that magnesium sulphate was just as beneficial as dexmedetomidine in reducing the hemodynamic pressor response brought on by laryngoscopy and endotracheal intubation.

A study to determine the efficacy of Dexmedetomidine as attenuator for hemodynamic response due to intubation was conducted by Kolkar et al., in the year 2015. As an outcome of the study the authors have concluded Dexmedetomidine at a dose of 0.6 mcg kg^{-1} premedication was able to attenuate the pressor response associated with laryngoscopy and tracheal intubation.

Nidhi B et al., (2016) conducted a study by enrolling 80 patients divided into 4 groups and treated them with different doses of intravenous Magnesium sulphate (Gr. I: 30 mg kg^{-1}; Gr. II: 40 mg kg^{-1}; Gr. III: 50 mg kg^{-1}; and Gr. IV: Control). From the study the authors had concluded Magnesium sulphate at a dose of <50mg kg^{-1} effectively in stabilizing the cardiovascular instability related to intubation.

In the same year 2015, Sridhar Kalkeri and his colleagues have investigated the effect of Dexmeditomidine on decreasing the hemodynamic pressor response due to intubation in patients undergone CABG. Administration of Dexmedetomidine to patients at a dose of 1 mcg kg^{-1} preoperatively was able to effectively obtrude the hemodynamic stress during intubation compared to the placebo control group of patients. The authors have concluded that Dexmedetomidine could be used regularly in the intensive care unit as an alternative to other drugs used.

A prospective, single-blinded, randomised, controlled clinical trial was carried out in 2016 by Rashi Kadam et al. to evaluate the efficacy of magnesium sulphate on the haemodynamic alteration during intubation. Group A got the same quantity of ordinary saline (over 5 min, 5 min prior to induction). Group B was given intravenously 30 mg kg-1 of magnesium sulphate (over 5min, 5 min prior to induction). The parameters of hemodynamics were noted. Because of its characteristics, such as greater effectiveness and fewer adverse effects, magnesium sulphate in a single bolus dosage can be used often to decrease the haemodynamic pressor response after intubation.

Dexmedetomidine's impact on intubation was studied by Arun Kumar et al in 2019. Fentanyl and Midazolam were given as premedicating to all of the patients. Dexmedetomidine 1mcg kg-1 was given to the study group over the course of 15 minutes in 100ml of saline, whereas normal saline was given to group C. The heart rate, systolic blood pressure, mean arterial pressure, and diastolic blood pressure were all considerably lower in the dexmedetomidine group than in the control group when administered at a dosage of 1 mcg kg-1 during intubation.

Ushasaha et al., in the year 2003 have investigated the pressor response and hypertension in their study. The stress response for laryngoscopy and intubation was measured in 128 hypertensive individuals who were scheduled for elective surgery under general anaesthesia. Previously, it was assumed that stopping antihypertensive medication before a planned surgery was riskier than the hypertension itself. However, new research suggests that continuing medications during the

perioperative period is beneficial. In normal persons, sympathetic activation causes a pressor reaction to laryngoscopy and intubation. Because the sympathetic nervous system is hypersensitive in hypertensive individuals, laryngoscopy and intubation produce an increased pressor reaction. As a result, there is a considerable danger of consequences, necessitating the use of methods or pharmacological drugs to soften the response.

Pharmacological treatments for reducing morbidity linked with pressor response of intubation were studied by Khan et al., 2013. The investigation was conducted against the backdrop of the use of numerous medicines to reduce or attenuate the cardiovascular response after intubation. The modifications caused did not cause much concern in normally healthy people, but they did cause some mortality and morbidity in high-risk patients. When comparing the risk of arrhythmia occurring during intubation with the use of local anaesthetics, beta blockers, calcium channel blockers, and opioids to the use of placebo, this analysis found that the risk of arrhythmia occurring during intubation was substantially lower with the use of these drugs. In addition, there is less evidence of ischemia in the ECG.

MATERIAL AND METHOD

Study design: Hospital based prospective, randomized, single blinded (Patient Blinded), comparative study.

Study Place: The study was conducted between the period of August 2021 and July 2022 at the Department of Anaesthesiology, Santosh Deemed to be University, Ghaziabad, UP.-

Inclusion Criteria:

- Patients of either sex, aged between 18 - 60 years.
- Patients belonging to American Society of Anaesthesiologists Class I & II and

Mallampatti grade I & II.

- All patients posted for surgery under general anaesthesia (GA).

Exclusion Criteria:

- Patient refused for the procedure.
- Patients with history of allergy or contraindications to either Dexmedetomidine or

Magnesium sulfate.

- Those with predicted difficulty in intubation, pregnant and nursing women, and morbid obesity, Coronary artery disease, ischemic heart disease, heart blocks, Diabetes mellitus and Patients with heart rate < 60 bpm and systolic blood pressure < 100 mmHg.

Study Population: Selection of patients for the current study was strictly adhered with the inclusion criteria. Patients undergoing elective surgical procedure under General Anaesthesia with controlled ventilation were taken up for study. Those who are not suitable for the inclusion criteria of this study were excluded to participate in the current study. Ethical clearance for the study has been obtained from the IEC of Santosh Deemed to be University, Ghaziabad & Informed Patient Consent has been taken from the study participants before the selection of patients to start this study.

Methodology:

A total of 64 patients from either sex belonging to ASA grade I and II admitted at Santosh Medical College and Hospital, Santosh Deemed to be University, Ghaziabad for elective surgery and those who are strictly suitable for the study with all the inclusion criteria were selected for the study. Before the start of the procedure the patients and randomly assigned into two groups namely, Group 1 & Group 2 respectively.

Pre-Anaesthetic Check-up: "A thorough physical and pre-anaesthetic examination was done a day before the surgery. All routine haematological, biochemical & radiological investigations, electrocardiogram appropriate for the surgery was done.

Anaesthesia Procedure:

- After 5 minutes of stabilisation of patient in operation theatre baseline heart rate (HR), systolic (SBP), diastolic (DBP), mean arterial pressure (MAP) were recorded.
- GROUP 1: Injected with Magnesium sulphate 30mg kg-1 diluted in 10 ml of normal saline intravenously over 10 minutes.
- GROUP 2: Injected with Dexmeditomidine 1mcg kg-1 diluted in 10 ml of normal saline intravenously over 10 minutes.
- All the patients in both the groups were preoxygenated and premedicated with standard general anaesthesia procedure i.e., with intra venous injections of Fentanyl 2mcg kg-1 with Propofol 2 mg kg-1 and Vecuronium 0.1mg kg-1. O2 and N2O were maintained with sevoflurane/isoflurane on IPPV. Haemodynamic parameters of patients including HR, SBP and DBP was recorded immediately after endotracheal intubation (ETI) and at 2, 5, 10 minutes after ETI.

Definitions followed based on baseline values:

- Tachycardia - HR>25% of the baseline value.
- Bradycardia - HR<20% of the baseline value.
- Hypotension - SBP $\leq$20% of the baseline value.

Observations:

- Baseline SBP, DBP, MAP.
- Baseline HR.
- SBP, DBP, HR every minute from 0 to 5 after intubation.
- Duration of surgery.
- Any complication.

Statistical Analysis:

The data was collected and entered in MS excel 2010. Different statistical analysis was performed using R software version 4.0.2. The one-sample Kolmogorov – Smirnov test was employed to determine whether the data sets differed from a normal distribution or not. Normally distributed data were analysed using parametric tests and non - normally distributed data was analysed using non parametric tests. Descriptive statistics was calculated for qualitative and categorical variables. Graphical representation of the variable is shown to understand the results clearly and to measure the association for categorical dataset were analysed using ANOVA. Independent T-test or student t-test was applied to measure the mean difference between two groups. Correlation was estimated to measure the strength of relationship between two or more quantitative variables. If p value <0.05, it were considered as statistically significant and if p-value >0.05, then it is statistically insignificant.

OBSERVATION AND RESULTS

Table 1: Average Age of Patients in the Study Groups

	Age	SD	
Group 1	**36.6176471**	**10.58305**	**P = 0.4746**
Group 2	**38.5588235**	**11.6416**	

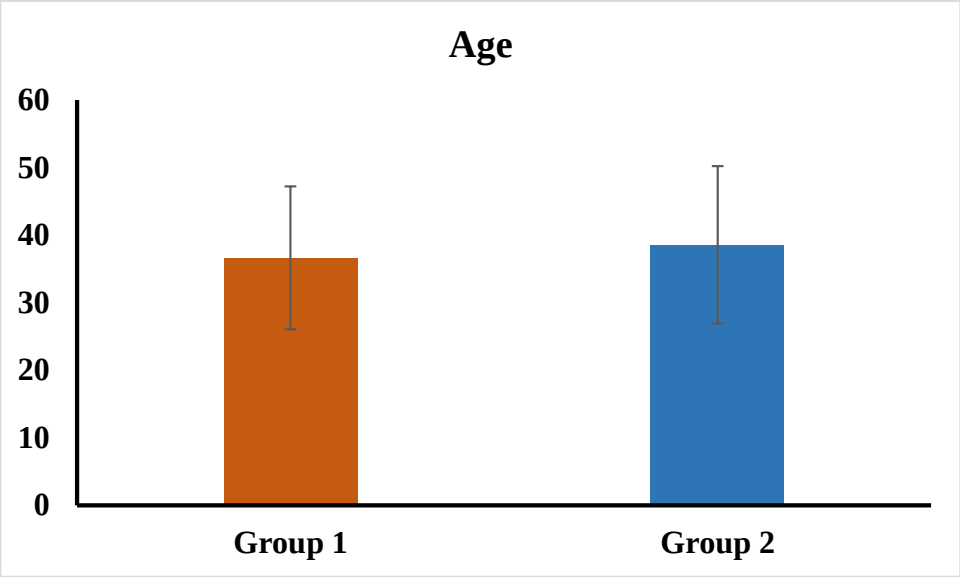


Table 2: Distribution of Gender in the Study Groups

Group 1	**Male**	**16**
	Female	**18**
Group 2	**Male**	**20**
	Female	**14**

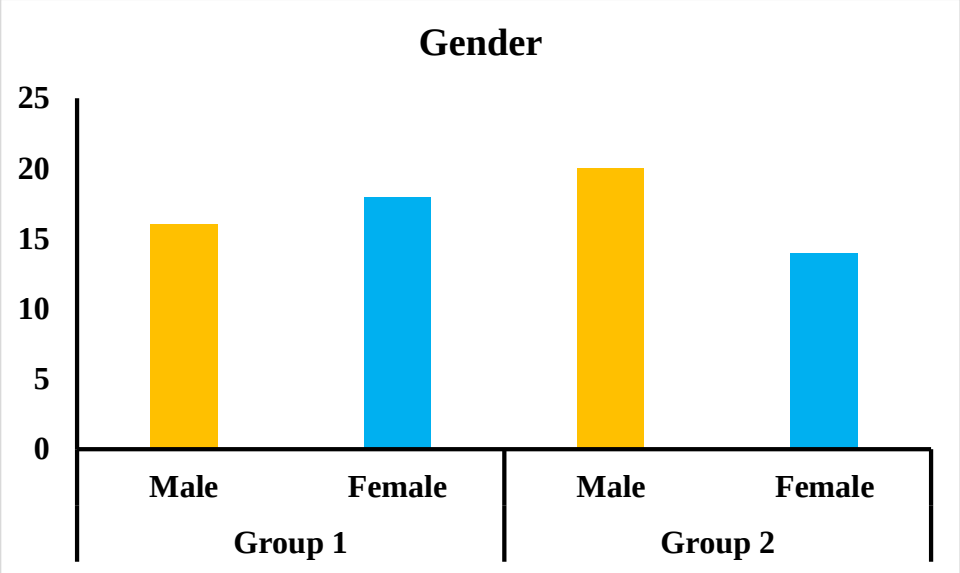


Table 3: Average Body Weight of the Patients in the Study Groups

		Weight	**SD**	
Body Weight	**Group 1**	**62.82**	**10.76039**	
	Group 2	**64.06**	**9.098589**	**P = 0.6096**

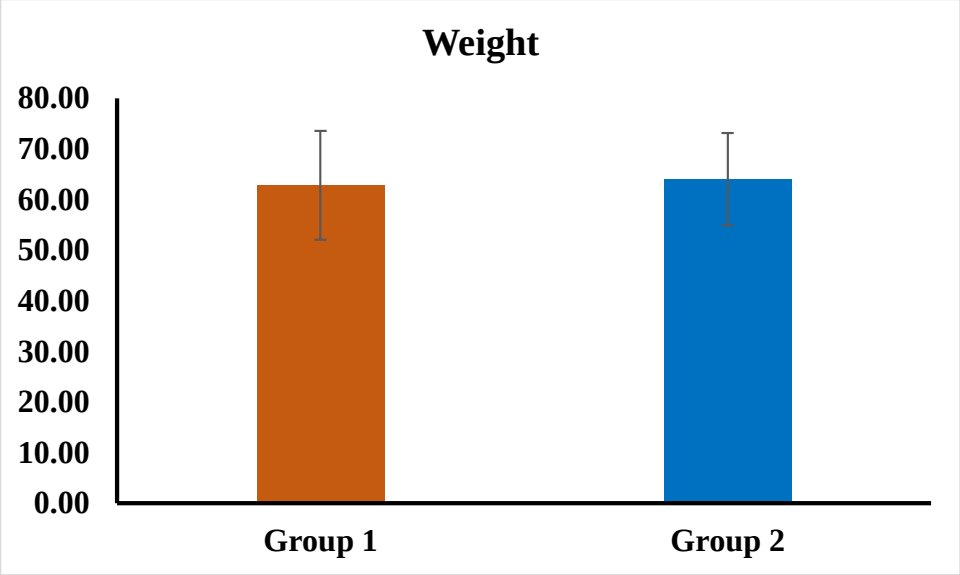


Table 4: Distribution of Patients in the Study Groups based on ASA Grades I & II

ASA I & II	**Group 1**	**ASA I**	**23**
		ASA II	**11**
	Group 2	**ASA I**	**22**
		ASA II	**12**

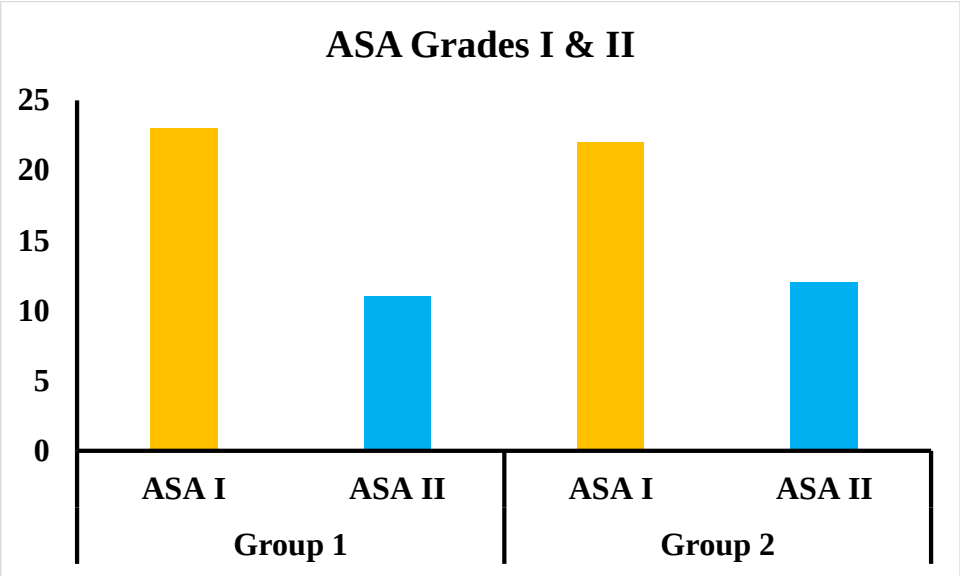


Table 5: Systolic Blood Pressure at different times points

	Group 1		Group 2		
Base line	**129**	**11.35**	**132**	**12.07**	**P = 0.2468**
0 Minute	**151**	**14.59**	**144**	**13.48**	**P = 0.0596**
1 Minute	**143**	**12.96**	**136**	**12.08**	**P = 0.0179**
2 Minutes	**135**	**13.59**	**129**	**9.60**	**P = 0.0287**
3 Minutes	**129**	**9.40**	**124**	**8.10**	**P = 0.0358**
4 Minutes	**125**	**8.08**	**121**	**4.23**	**P = 0.0261**
5 Minutes	**121**	**6.85**	**117**	**2.39**	**P = 0.0009**

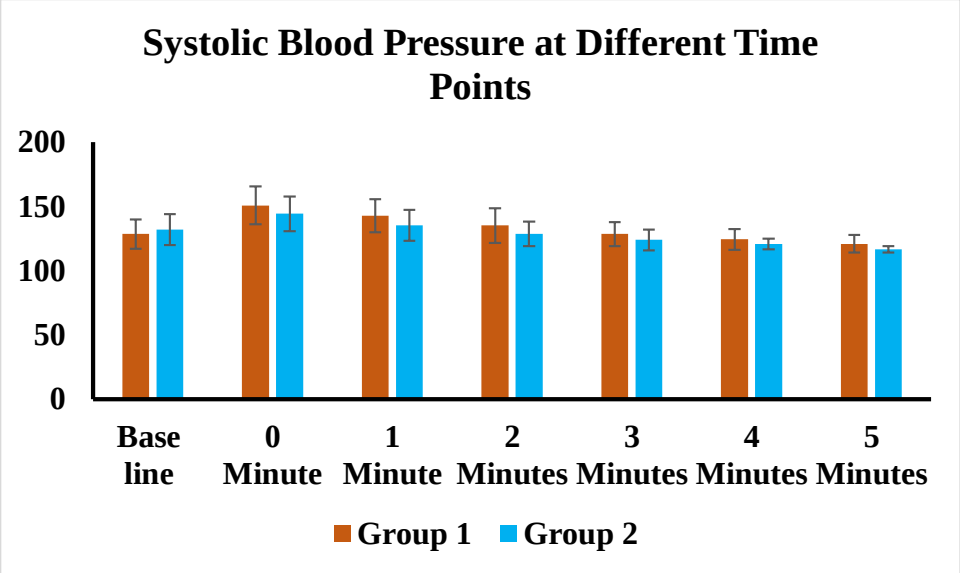


Table 6: Diastolic Blood Pressure at different times points					
	Group 1		**Group 2**		
Base line	**79**	**6.04**	**82**	**4.57**	**P = 0.0503**
0 Minute	**90**	**9.92**	**91**	**7.31**	**P = 0.7918**
1 Minute	**85**	**7.72**	**86**	**6.93**	**P = 0.6911**
2 Minutes	**81**	**6.96**	**82**	**6.15**	**P = 0.2897**
3 Minutes	**78**	**6.73**	**79**	**5.68**	**P = 0.5102**
4 Minutes	**75**	**5.91**	**74**	**6.61**	**P = 0.4444**
5 Minutes	**72**	**5.42**	**67**	**6.32**	**P = 0.0005**

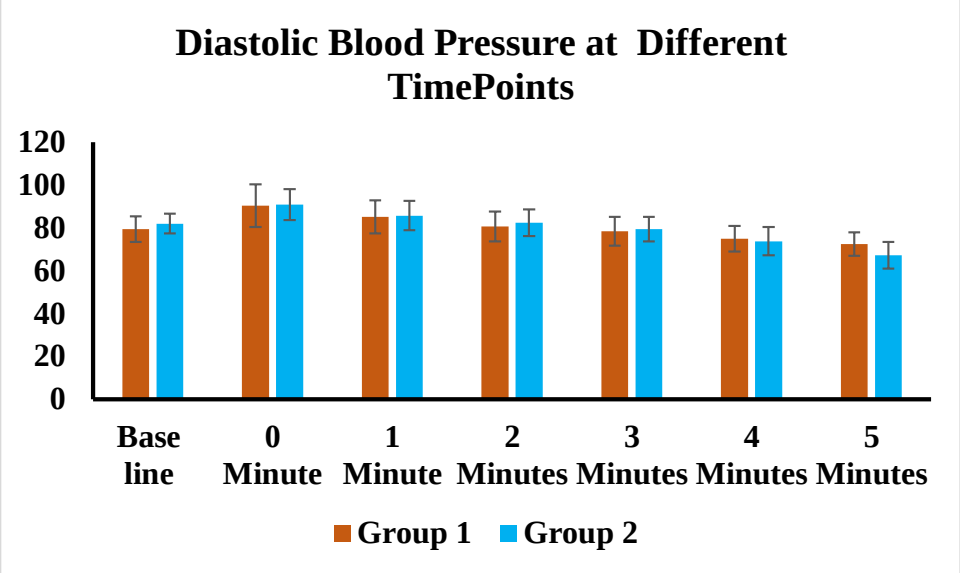


Table 7: Mean Arterial Pressure at different times points					
	Group 1		Group 2		
Base line	96	6.71	99	6.48	P = 0.0496
0 Minute	110	9.81	109	7.77	P = 0.5971
1 Minute	104	7.08	102	6.77	P = 0.2404
2 Minutes	99	6.37	98	5.67	P = 0.5008
3 Minutes	95	5.86	94	4.98	P = 0.5166
4 Minutes	91	5.45	89	4.45	P = 0.1073
5 Minutes	89	4.98	84	4.13	P < 0.0001

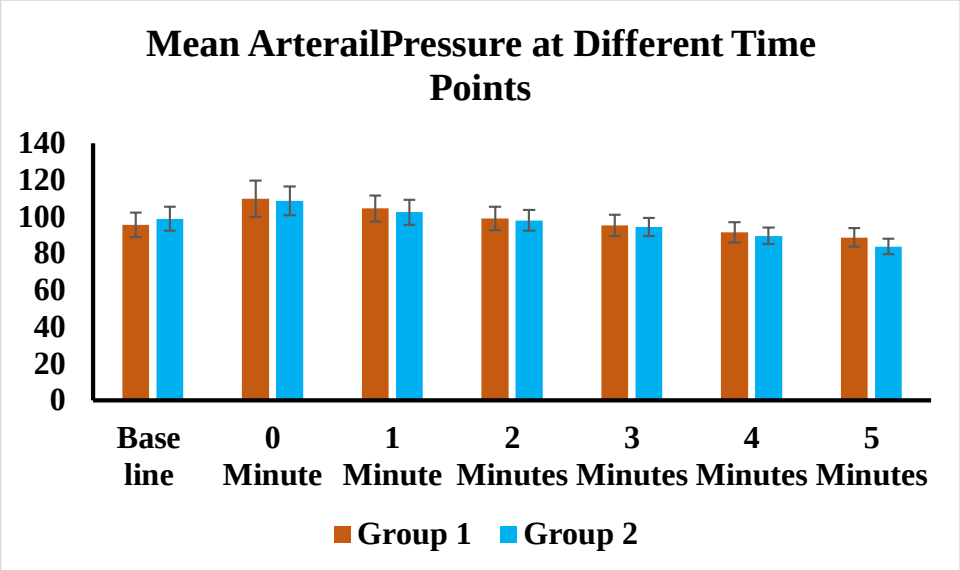
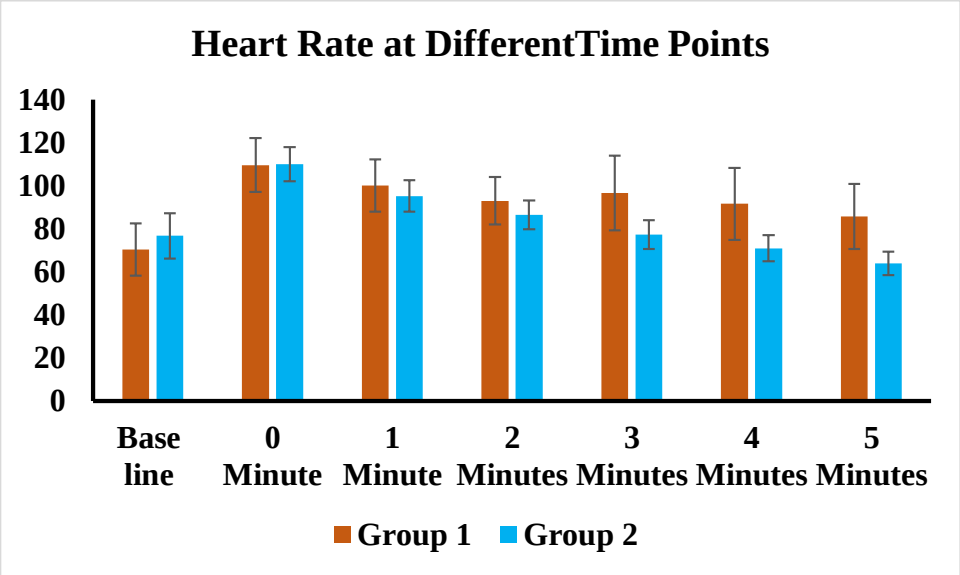


Table 7: Heart Rate at different times points					
	Group 1	SD	Group 2	SD	
Base line	70	12.17	77	10.56	P = 0.0228
0 Minute	110	12.47	110	8.00	P = 0.9002
1 Minute	100	12.09	95	7.33	P = 0.0510
2 Minutes	93	10.98	86	6.72	P = 0.0043
3 Minutes	97	17.35	77	6.63	P < 0.0001
4 Minutes	92	16.81	71	6.12	P < 0.0001
5 Minutes	86	15.09	64	5.53	P < 0.0001



DISCUSSION

Hemodynamic reaction to laryngoscopy has been a heavily debated subject since 1940. These reactions may deteriorate in elderly individuals and patients with hemodynamic compromise as a result of elevated blood pressure, heart rate, and oxygen consumption. Even patients with normal blood pressure can experience hypertension and tachycardia during laryngoscopy, thus it is surprising that problems are not more frequent, perhaps due to their temporary nature. Controlling this perioperative stress response is therefore a key objective of anaesthesia.

Hemodynamic reaction to laryngoscopy has been a heavily debated subject since 1940. These reactions may deteriorate in elderly individuals and patients with hemodynamic compromise as a result of elevated blood pressure, heart rate, and oxygen consumption. Even patients with normal blood pressure can experience hypertension and tachycardia during laryngoscopy, thus it is surprising that problems are not more frequent, perhaps due to their temporary nature. Controlling this perioperative stress response is therefore a key objective of anaesthesia.

In order to examine the effectiveness of dexmedetomidine and magnesium sulphate for attenuating hemodynamic stress response during laryngoscopy and endotracheal intubation, we organised a prospective, randomised, double-blind trial at Santosh Medical College and Hospital, Ghaziabad, UP. In this study, 68 ASA physical status I and II patients between the ages of 18 and 60 who were scheduled for elective procedures were included. Group D (Dexmedetomidine group) and Group M , the two study groups, were assigned to them at random .

Patient Characteristics across the group:

There was no statistically significant difference between the two groups' age, weight, and ASA status, which are all demographic factors. The patients were equivalent in both groups and belonged to either ASA I or ASA II.

Dosage of the drug: Magnesium sulphate 30 mg/kg in 100 ml of normal saline was used in group 1 of the current investigation, while dexmedetomidine 1 mcg/kg diluted in 100 ml of normal saline was administered in group 2. Ten minutes prior to intubation, the medication was administered over the course of 15 minutes.

Magnesium sulphate 30 mg/kg and dexmedetomidine 1 mcg/kg were employed in the study conducted by Krishna Chaitanya. Although both medications were successful at lowering blood

pressure, dexmedetomidine was better at lowering heart rate. Both groups of patients had stable hemodynamics. Both groups experienced uneventful recovery. Similar research was done by Rashi Kadam et al., who employed 30 mg/kg of magnesium sulphate and compared it to normal saline. During and following induction, the magnesium group's systolic, diastolic, and heart rate readings all considerably decreased. Extubation and recovery took longer in the research group.

Different dosages of magnesium sulphate were used in a 2013 study by Nidhi B et al. group one: 30 mg/kg, group two: 40 mg/kg, and group three: 50 mg/kg The prevalence of hypotension is higher in group 3 (50 mg/kg magnesium sulphate). Magnesium sulphate (10, 20, 30, 40, or 50 mg/kg) was given to the patients in five groups by K. Montazeri et al five minutes prior to induction. When compared to the other groups, group C (30 mg/kg of magnesium sulphate) saw reduced variation in heart rate and mean arterial pressure ($p > 0.05$).

Magnesium sulphate was administered by Gautham Pillai et al. as a 30 mg/kg bolus before induction and a 10 mg/kg/hr continuous infusion. The M group's hemodynamic parameter was noticeably lower. Kalkeri et al did a study in which Dexmedetomidine 1 mcg/kg body weight dissolved in 100 ml of normal saline was administered over a 15-minute period before induction to considerably inhibit the haemodynamic stress responses to intubation without having any notable adverse effects, such as hypotension and bradycardia. Similar to this, Kolkar et al. administered intravenous Dexmedetomidine at a dose of 0.6 mcg/kg to reduce tension during intubation. After laryngoscopy and intubation, attenuation begins within 5 minutes and reaches its peak within 10 minutes. After 5 to 10 minutes following intubation, the hemodynamic parameters are close to their baseline levels. Similar to this, Nabin porkhel et al administered intravenous Dexmedetomidine 1 mg/kg to reduce the intubation stress reaction.

Dexmedetomidine was used by Rajdip Hazra et al. at a dosage of 0.5 mcg/kg to reduce the cardiovascular stress response. In our investigation, we steered clear of the infusion of Magnesium sulphate and Dexmedetomidine. Saraf et al. investigated the effectiveness of 0.6 mcg/kg Dexmedetomidine to blunt the pressor response of tracheal intubation. In comparison to Montazeri et al., who employed 50mg/kg of magnesium sulphate, we utilised 30mg/kg, which exhibited a lower incidence of hypotension and sedation. Dexmedetomidine was utilised at a dosage of 1mcg/kg, which had a favourable impact with a lower incidence of side effects.

Hemodynamic stability: Several studies have shown that the administration of intravenous magnesium sulphate and dexmedetomidine for laryngoscopy and intubation causes a cardiovascular stress response. During the following intervals: baseline, during intubation, 1

minute after intubation, 2 minutes after intubation, 3 minutes after intubation, 4 minutes after intubation, and 5 minutes after intubation, the variation in hemodynamic parameters such as heart rate, systolic blood pressure, diastolic blood pressure, and mean arterial blood pressure was noted.

Heart Rate: Dexmedetomidine was shown to lower heart rate more than magnesium sulphate, according to research by Krishna Chaitanya et al. When compared to Magnesium sulphate, Dexmedetomidine showed significantly lower heart rate values (p 0.05). The mean heart rate in the magnesium sulphate group did not vary by more than 10 beats per minute, and the heart rate reverted to normal within 5 minutes, despite the fact that the p value for heart rate is statistically significant. The outcomes matched those of our research.

Magnesium sulphate was employed by Gautham Pillai et al. in a comparison study with normal saline, using a 30 mg/kg bolus before induction and a 10 mg/kg/hr continuous infusion. Just after intubation, Group M had considerably reduced mean arterial pressure and heart rate (p 0.05). This is not the case with our research. As they employed continuous infusion throughout the research, bradycardia was seen. Therefore, we did not use continuous infusion for this investigation.

Kalkeri et al. used 1 mcg/kg of dexmedetomidine. The D group's heart rate was significantly reduced. The outcomes were equivalent to those of our study.

Kolkar et al. employed 0.6 mcg/kg iv Dexmedetomidine, which statistically significantly suppressed mean heart rate more effectively than the control group. The outcomes were equivalent to those of our study.

Dexmedetomidine 0.6 mcg/kg was employed by Saraf et al, who observed a drop in heart rate in group D. The outcomes were equivalent to those of our study. Magnesium sulphate, 30 mg/kg bolus and 10 mg/kg/hr continuous infusion, were employed by Gautham Pillai et al. to compare to regular saline. In Group M, the heart rate was substantially lower (p 0.05). When compared to the magnesium sulphate group in our investigation, the dexmedetomidine group demonstrated a statistically significant drop in heart rate (P 0.05).

Systolic blood pressure: Using Magnesium sulphate 30 mg/kg and Dexmedetomidine 1 mcg/kg, Krishna Chaitanya et al. found a significant difference between the medications at 0 minutes, 1, 3, and 5 minutes for systolic blood pressure (p 0.005). The outcomes matched those of our research. In a randomised control trial by Rajan Sunil et al, participants in group B received 1.5 mg/kg of lignocaine 90 seconds before intubation in addition to 50 mg/kg of intravenous

magnesium sulphate. There was little distinction between the groups. The outcomes matched those of our research.

Magnesium sulphate 30mg/kg was used by Rashi Kadam et al five minutes before induction. In the first five minutes following intubation, the study group's average systolic blood pressure was noticeably low. The difference in systolic blood pressure was statistically significant since he used a placebo as a control, which is consistent with the findings of our study.

Dexmedetomidine 1 mcg/kg was used by Arun Kumar Patra et al. In the first five minutes following intubation, the study group's average systolic blood pressure was statistically significant lower than normal. This was consistent with our research.

Dexmedetomidine 1mcg/kg was administered 5 min before induction by Kalkeri et al, with normal saline serving as the control. When compared to the control group, he saw a statistically significant drop in blood pressure. This was different from our study since they utilised normal saline as a control, which was why.

Dexmedetomidine 0.5 mcg/kg was administered 5 min before induction by Rajdip Hazara et al, with normal saline serving as the control. When compared to the control group, he saw a statistically significant drop in blood pressure. This was not the case with our research.

In our investigation, statistically significant differences in systolic blood pressure between the two groups were seen at different time points.

Magnesium sulphate 30 mg/kg and dexmedetomidine 1 mcg/kg were employed by Krishna Chaitanya et al to measure diastolic blood pressure; however, there was no statistically significant difference between the two medicines at 0, 1, 2, 3, and 4 minutes ($p > 0.005$). The outcomes matched those of our research.

In a randomised control trial conducted by Rajan Sunil et al, participants in group A got 1.5 mg/kg of lignocaine 90 seconds before intubation, whereas those in group B received iv magnesium sulphate 50 mg/kg body weight given 10 min before induction. Diastolic blood pressure decreased after induction, but there was no discernible difference between the groups. The outcomes matched those of our research. Magnesium sulphate 30mg/kg was utilised by Rashi Kadam et al five minutes before induction. In the first five minutes following intubation, the average diastolic blood pressure in the study group was considerably lower than in the control group (p.005). Dexmedetomidine was utilised by Saraf et al at 0.6 mcg/kg, and he saw a substantial drop in diastolic blood pressure compared to the control group. Dexmedetomidine 1

mcg/kg was used by Arun Kumar Patra et al. In the first five minutes following intubation, the average diastolic blood pressure in the study group was considerably lower than in the control group, which was statistically significant. This was not the case with our research.

The cause may be because the control, normal saline, has no effect on regulating cardiovascular stress reactions to laryngoscopy and endotracheal intubation. Thus, they demonstrated a considerable rise in systolic, diastolic, and mean arterial pressure during laryngoscopy. No statistically significant differences in diastolic blood pressure were seen between the two groups at any point throughout our investigation.

Magnesium sulphate 30 mg/kg and dexmedetomidine 1 mcg/kg were employed by Krishna Chaitanya et al, and there was no statistically significant difference ($p > 0.005$) in the mean arterial pressures for the two medications at intervals of 0, 1, 2, 3, and 4. The outcomes matched those of our research.

A randomised control trial was conducted by Rajan Sunil et al. 10 minutes prior to induction, he administered 50 mg/kg of magnesium sulphate intravenously, while group B recipients got 1.5 mg/kg of lignocaine 90 seconds before intubation. Mean arterial pressure decreased after induction at intervals of 0, 1, 3, 5, and 10 minutes, but there was no discernible difference between the groups. The outcomes matched those of our research.

Magnesium sulphate 30mg/kg was used in the study by Salman et al, who found a significant difference in mean arterial blood pressure between the magnesium and control groups. In contrast to our study, the outcome.

In a randomised control research by K. Montazeri et al, magnesium sulphate 30 mg/kg body weight was administered intravenously 10 min before the patient was induced, and the mean arterial blood pressure between the two groups differed significantly between the groups. In contrast to our study, the outcome.

Dexmedetomidine 0.6 mcg/kg was used by Kolkar et al. He noticed a considerable drop in the mean blood pressure compared to the control group.

Dexmedetomidine was used by Saraf et al at 0.6 g/kg, and he saw a substantial drop in mean arterial pressure compared to the control group. This was not the case with our research.

With the exception of the first five minutes of our investigation, there were no statistically significant differences in mean arterial pressure between the two groups.

SUMMARY & CONCLUSION

At Thanjavur Medical College Hospital, Thanjavur, we performed a prospective double-blinded randomised control trial on 68 patients having elective noncardiac procedures. Patients between the ages of 18 and 60, of both sexes, were included. In order to lessen the cardiovascular stress reactions to laryngoscopy and endotracheal intubation, we compared the effects of dexmedetomidine 1 mcg/kg and magnesium sulphate 30 mg/kg given intravenously.

Using the closed cover approach, patients were randomly split into two groups of 34 each. Magnesium sulphate 30 mg/kg in 100 ml of normal saline was given to Group M. Dexmedetomidine 1 g/kg in 100 ml of normal saline was administered to Group D. It took 15 minutes and 10 minutes before induction to administer the test drug. Systolic, diastolic, and mean arterial pressure as well as the heart rate were recorded in both groups.

The nominal data were analysed using One-Way ANOVA, while the categorical data were evaluated using the Chi Square test. The mean and standard deviation are used to express the data, which were acquired as a range. It was deemed statistically significant when the probability value, or "p," was less than 0.05. In our research, we found that dexmedetomidine reduced heart rate and systolic blood pressure more than magnesium sulphate (p 0.005).

In our investigation, there was no statistically significant difference in mean arterial pressure or diastolic blood pressure between those groups.

Therefore, based on the findings of the present investigation, it can be said that dexmedetomidine 1 mcg/kg and magnesium sulphate 30 mg/kg intravenously supplied are both equally efficient for reducing hemodynamic stress responses to laryngoscopy and endotracheal intubation.

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ANNEXURE 1

PROFORMA

1. GENERAL INFORMATION

Name	
Age (in years)/ Sex	
Occupation	
Address	
Diagnosis	
Operation proposed	
MRD No.	
Surgery Unit	
Date	

2. GENERAL EXAMINATION

General Condition	
Any positive history	
Heart Rate(beats per minute)	
Blood Pressure(in mm of Hg)	
Temperature (in degree Fahrenheit)	
Spo2 (in %)	
Mouth Opening	
MPG grade	
Neck movement	
Oral examination	
ASA Grade	

3. SYSTEMIC EXAMINATION:

System	**Findings**
Respiratory system	
Cardiovascular system	
Central nervous system	
Per abdomen(G.I system)	

4. ANY SYSTEMIC DISEASE:

5. INVESTIGATIONS:

6. DEMOGRAPHIC PARAMETERS:

Parameters	
Age	
Weight (BMI)	
Height	
Duration of surgery	

7. GROUP ALLOCATON:

GROUP M:	
GROUP D:	

12 .HEMODYNAMIC CHANGES:

Time	Group M				Group D			
Baseline	HR	SBP	DBP	MAP	HR	SBP	DBP	MAP
Before intubation								
1 min. after intubation								
2 min. after intubation								
3 min. after intubation								
4 min. after intubation								
5 min. after intubation								

ANNEXURE 2

DEPARTMENT OF ANAESTHESIA SANTOSH MEDICAL COLLEGE & HOSPITAL GHAZIABAD, U.P

I am being requested to take part in a study entitled.

"COMPARATIVE STUDY OF INTRAVENOUS DEXMEDETOMIDINE AND INTRAVENOUS MAGNESIUM SULPHATE TO ATTENUATE HEMODYNAMIC STRESS RESPONSE TO LARYNGOSCOPY AND INTUBATION"

VOLUNTARY PARTICIPATION

My participation in the study is entirely voluntary and I am free to with draw from the study as and when I feel

Name ----------

Signature ----------

INFORMED CONSENT FORM

I _____ son/daughter/wife of _____ resident of_____ give my full, free and voluntary consent to be included as a subject in the study entitled "COMPARATIVE STUDY OF INTRAVENOUS DEXMEDETOMIDINE AND INTRAVENOUS MAGNESIUM SULPHATE TO ATTENUATE HEMODYNAMIC STRESS RESPONSE TO LARYNGOSCOPY AND INTUBATION" I have been explained, in my own language, and to my full satisfaction, the aim and nature of the study and risks and benefits. I have also been explained that my confidentiality will be maintained and all the investigations/ interventions will be carried out only after my consent is obtained. I am aware of my right to opt out of the study at any point without giving any reason, and without penalty or loss of routine care benefits.

Patient's/ Patient's relative's signature Doctor's Signature:

Or thumb impression.

Name: Date:

We have witnessed that the patient signed the above form in the presence of his free will after fully understanding its content.

Signature of Witness Signature of Investigator

Name: Name:

ANNEXURE 3

संतोषमेडिकलकॉलेजएवंअस्पताल (गाजियाबाद)

सूचितसहमति

मैं_____बेटा_____निवासी_____
____ घोषणा करता हूं कि मैं डॉ._____, ने मेरी भाषा में अपने अध्ययन, अध्ययन के उद्देश्य के बारे मे, और अध्ययन के दौरान अपनाई जानेवाली विभिन्न प्रयोगशाला जांच के बारे में सूचित कर दिया है। मुझे अध्ययन की अवधि और उस के दौरान अपनाए जानेवाले निर्देश भी मेरी भाषा मे समझा दिए गए हैं| मैं उपरोक्त अध्ययन में बिना किसी दबाव के दाखिला ले रहा हूं और पूर्ण सहमति दे रहा हूं| सन्तोषअस्पताल, गाजियाबाद में अपने आगे के उपचार पर बिना प्रतिकूल प्रभाव डाले इस अध्ययन से और प्रयोगशाला जांच की प्रकृति से मैं जब चाहूँ बाहर जाने के अपने अधिकार को सुरक्षित रख रहा हूं I

रोगी / रोगी के रिश्तेदार के हस्ताक्षर डॉक्टर के हस्ताक्षर या अंगूठे का निशान

नाम

नांक:

मेरी उपस्थिति में मरीज ने पूरी तरह से डाकटर के बताए गए अध्ययन और निर्देशों को समझने के बाद अपनी स्वतंत्र इच्छा से ऊपरके फार्म पर हस्ताक्षर कि ए हैं

Signature of Investigator

ANNEXURE 4

LIST OF ABBREVIATIONS

1. LETI – LARYNGOSCOPY AND ENDOTRACHEAL INTUBATION
2. ETT – ENDOTRACHEAL TUBE
3. SD – STANDARD DEVIATION
4. LA – LOCAL ANESTHETIC
5. NSAID- NON STEROIDAL ANTI INFLAMMATORY DRUGS
6. CO2 – CARBON DIOXIDE
7. IM- INTRA MUSCULAR
8. I V – INTRA VENOUS
9. CNS – CENTRAL NERVOUS SYSTEM
10. HR- HEART RATE
11. SBP – SYSTOLIC BLOOD PRESSURE
12. DBP – DIASTOLIC BLOOD PRESSURE
13. MAP – MEAN ARTERIAL PRESSURE
14. DOA- DURATION OF ANALGESIA
15. SPO2 – OXYGEN SATURATION
16. ETCO2 - END TIDAL CARBON DIOXIDE
17. ASA – AMERICAN SOCIETY OF ANAESTHESIOLOGY

ANNEXURE 5

ETHICAL COMMITTEE CLEARANCE

SANTOSH
Deemed to be University
(Established u/s 3 of the UGC Act, 1956)

F. No. SU/2021/1830[40] Dated: 17.07.2021

Subject: Grant of Ethical Clearance for Thesis Protocol Topic **"COMPARATIVE STUDY OF INTRAVENOUS DEXMEDETOMIDINE AND INTRAVENOUS MAGNESIUM SULPHATE TO ATTENUATE HEMODYNAMIC STRESS RESPONSE TO LARYNGOSCOPY AND INTUBATION.",** submitted by **Dr. Priyanshi Verma,** MD/MS student of Batch 2020-2021, Department of **Anaesthesia**.

*****

With reference to his/her request for grant of Ethical Clearance for the Thesis Protocol entitled **"COMPARATIVE STUDY OF INTRAVENOUS DEXMEDETOMIDINE AND INTRAVENOUS MAGNESIUM SULPHATE TO ATTENUATE HEMODYNAMIC STRESS RESPONSE TO LARYNGOSCOPY AND INTUBATION.", Dr. Priyanshi Verma** is informed that the Thesis Protocol submitted by him/her was considered by the **Screening Committee** of the Santosh Medical College & Hospital in its meetings held on 19.03.2021, 22.03.2021, 24.03.2021, 25.03.2021, 26.03.2021, 27.03.2021 and 06.04.2021. The recommendations of the **Screening Committee** were considered in detail by the Institutional **Ethics Committee** in its meeting held on **12.07.2021** and the same was **approved** by the **Ethics Committee**.

He /She is informed accordingly for further necessary action.

[Dr. V. P. Gupta]
REGISTRAR

Dr. Priyanshi Verma
MD/MS student of batch 2020-2021
Department of Anaesthesia
Email: vermapriyanshi@gmail.com

Copy to:

1. The Vice Chancellor
2. The Dean, SMC&H
3. The Dean – Research
4. The Dean – Academics
5. The Director - IQAC
6. Dr. Suveer Sharma (Guide)
7. HOD of **Anaesthesia**

ANNEXURE 6

PLIAGARISM CERTIFICATE

Ouriginal by Turnitin

Document Information

Analyzed document	Dr. Priyanshi Thesis - v1-20102022 (1).docx (D149321651)
Submitted	2022-11-11 06:24:00
Submitted by	Dr Jyoti Batra
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W	URL: https://www.researchgate.net/publication/289693697_Comparison_of_hemodynamic_responses_to_lary... Fetched: 2019-10-07 09:24:46	1

DIAGNOSIS	SURGERY	AGE/SEX	Age	Gender	WEIGHT (KG)	ASA	DRUG USED	BASELINE SBP	BASELINE DBP	BASELINE MAP	BASELINE HR	SBP 0 MIN	DBP 0 MIN	MAP 0 MIN	HR 0 MIN	SBP 1 MIN	DBP 1 MIN	MAP 1 MIN	HR 1 MIN	SBP 2 MIN	DBP 2 MIN	MAP 2 MIN	HR 2 MIN	SBP 3 MIN	DBP 3 MIN	MAP 3 MIN	HR 3 MIN	SBP 4 MIN	DBP 4 MIN	MAP 4 MIN	HR 4 MIN	SBP 5 MIN	DBP 5 MIN	MAP 5 MIN	HR 5 MIN
Cholesystectomy	Lap chole	55/F	55	F	50	2	$MgSO_4$	157	88	111	78	160	80	112	79	157	73	101	78	143	66	92	76	122	61	81	73	113	60	78	70	106	59	75	70
Cholesystectomy	Lap chole	32/M	32	M	70	1	$MgSO_4$	110	80	90	84	151	98	90	103	142	84	103	92	142	78	99	91	117	72	87	86	114	68	83	87	112	69	83	87
Bladder tumor	TURBT	58/M	58	M	75	2	$MgSO_4$	132	86	101	88	158	110	126	108	154	108	123	106	146	102	117	100	140	98	112	98	138	90	106	96	134	86	102	90
Cholesystectomy	Lap chole	30/F	30	F	68	2	$MgSO_4$	130	90	103	90	204	108	140	120	180	90	120	98	178	78	111	74	134	70	91	69	124	70	88	61	124	74	91	56
CSOM right ear	Tympanoplasty	45/F	45	F	72	2	$MgSO_4$	148	76	91	62	169	77	100	101	138	78	98	71	130	70	90	76	120	66	84	74	111	63	79	72	110	62	78	68
Appendicitis	Appendectomy	35/F	35	F	65	1	$MgSO_4$	130	80	97	78	166	88	114	99	160	76	104	96	156	72	100	91	150	70	97	88	143	68	93	84	136	65	89	80
Cholesystectomy	Lap chole	44/F	44	F	60	1	$MgSO_4$	110	78	89	80	158	80	106	98	155	78	104	90	148	76	100	87	142	74	97	84	136	72	93	80	130	70	90	78
Cholesystectomy	Lap chole	30/M	30	M	75	1	$MgSO_4$	114	76	89	82	164	90	115	110	160	82	108	98	154	80	105	92	146	79	101	84	140	76	97	79	134	73	93	76
Cholesystectomy	Lap chole	45/M	45	M	70	2	$MgSO_4$	148	88	108	77	158	92	114	113	152	88	109	96	150	85	107	92	142	84	103	86	134	80	98	82	128	78	95	80
CSOM left ear	Tympanoplasty	50/F	50	F	55	2	$MgSO_4$	136	86	103	76	148	88	108	140	145	86	106	100	140	83	102	91	132	81	98	88	126	78	94	81	120	76	91	76
DNS to rt.	Sepstoplasty	35/F	35	F	50	1	$MgSO_4$	120	70	87	90	150	100	117	105	145	86	106	108	138	84	102	101	130	82	98	94	122	76	91	89	118	75	89	85
Cholesystectomy	Open chole	33/F	33	F	67	1	$MgSO_4$	124	78	93	92	140	110	120	120	142	90	107	110	132	86	101	103	126	82	97	98	121	77	92	94	116	73	87	90
Cholesystectomy	Lap chole	28/M	28	M	66	1	$MgSO_4$	130	82	98	82	152	105	121	112	150	94	113	98	142	84	103	90	134	81	99	86	126	74	91	82	122	72	89	76
CSOM right ear	Tympanomastoidectomy	25/F	25	F	48	1	$MgSO_4$	118	78	91	76	158	90	113	130	156	90	112	126	146	82	103	122	140	80	100	116	138	76	97	111	130	71	91	103
Cholesystectomy	Lap chole	55/M	55	M	60	2	$MgSO_4$	146	88	107	55	160	98	119	118	154	94	114	116	148	84	105	100	142	82	102	92	135	79	98	90	131	76	94	84
Cholesystectomy	Lap chole	44/F	44	F	60	2	$MgSO_4$	134	80	98	62	152	78	103	119	150	74	99	101	142	72	95	94	132	74	93	90	126	70	89	82	121	69	86	76
Fracture shaft of rt. ulna	ORIF rt. Ulna	22/M	22	M	70	1	$MgSO_4$	124	68	87	68	148	84	105	99	143	80	101	89	130	78	95	81	128	75	93	78	122	72	89	74	117	70	86	70
DNS to lt.	Septoplasty	23/M	23	M	68	1	$MgSO_4$	130	72	91	64	138	86	103	98	136	84	101	90	125	79	94	80	124	76	92	75	120	74	89	70	116	73	87	64
Cholesystectomy	Open chole	36/F	36	F	50	1	$MgSO_4$	118	70	86	61	140	90	107	111	134	88	103	100	122	86	98	92	120	84	96	86	116	80	92	82	118	78	91	74
Cholesystectomy	Lap chole	32/M	32	M	80	1	$MgSO_4$	120	80	93	58	150	102	118	140	140	90	107	132	128	89	102	123	123	86	98	115	121	84	96	110	120	83	95	100
Cholesystectomy	Lap chole	45/M	45	M	78	1	$MgSO_4$	122	70	87	64	160	98	119	115	148	96	113	110	136	90	105	101	132	85	101	126	126	82	97	120	122	80	94	112
Umbilical hernia	Mesh Hernioplasty	42/M	42	M	85	1	$MgSO_4$	126	66	86	55	162	78	106	112	150	74	99	98	138	73	95	90	135	76	96	130	127	74	92	124	123	72	89	118
Fracture mandible	ORIF mandible	22/M	22	M	65	1	$MgSO_4$	130	80	97	63	148	76	100	110	134	74	94	98	122	72	89	91	118	73	88	116	120	70	87	109	119	67	84	101
Thyroid mass	Radical thyroidectomy	30/F	30	F	48	1	$MgSO_4$	132	78	96	82	140	80	100	119	130	78	95	110	120	76	91	99	120	75	90	114	117	71	86	111	116	68	84	102
Cholesystectomy	Lap chole	31/F	31	F	50	1	$MgSO_4$	128	76	93	72	144	88	107	99	128	84	99	96	118	80	93	98	120	78	92	112	118	76	90	102	117	74	88	92
Cholesystectomy	Open chole	32/M	32	M	65	1	$MgSO_4$	138	82	101	50	162	86	111	100	144	86	105	90	138	81	100	90	134	79	97	124	130	72	91	120	126	73	91	111
Fracture shaft of rt.radius	ORIF lt. radius	26/F	26	F	45	1	$MgSO_4$	128	84	99	62	136	90	105	101	126	88	101	92	120	84	96	82	117	83	94	114	121	80	94	105	117	76	90	96
Cholesystectomy	Lap chole	43/F	43	F	50	1	$MgSO_4$	120	78	92	58	128	92	104	108	120	90	100	99	117	86	96	89	118	84	95	111	121	81	94	106	119	76	90	96
Cholesystectomy	Lap chole	50/M	50	M	70	2	$MgSO_4$	140	80	100	82	150	86	107	100	140	84	103	96	132	83	99	87	130	80	97	120	126	77	93	113	123	74	90	102
CSOM left ear	Tympanoplasty	20/M	20	M	66	1	$MgSO_4$	116	82	93	64	130	82	98	109	128	80	96	106	120	76	91	100	116	75	89	110	118	71	87	101	117	67	84	92
Umbilical hernia	Mesh Hernioplasty	35/F	35	F	56	1	$MgSO_4$	120	80	93	62	132	88	103	112	126	86	99	110	122	81	95	102	120	80	93	95	116	76	89	90	118	74	89	86
DNS to rt.	Septoplasty	22/F	22	F	49	1	$MgSO_4$	118	80	93	58	128	78	95	122	120	76	91	116	118	75	89	103	122	74	90	96	121	72	88	91	117	69	85	87
Cholesystectomy	Lap chole	46/F	46	F	55	2	$MgSO_4$	142	84	103	54	140	90	107	99	138	86	103	96	130	82	98	90	126	80	95	80	124	76	92	72	120	72	88	68
Cholesystectomy	Lap chole	44/M	44	M	75	2	$MgSO_4$	138	86	103	59	150	104	119	98	136	96	109	90	128	90	103	83	125	86	99	76	123	81	95	72	118	69	85	65

Chol: 19

DIAGNOSIS	SURGERY	AGE/SEX			WEIGHT (KG)	ASA	DRUG USED	BASELINE SBP	BASELINE DBP	BASELINE MAP	BASELINE HR	SBP 0 MIN	DBP 0 MIN	MAP 0 MIN	HR 0 MIN	SBP 1 MIN	DBP 1 MIN	MAP 1 MIN	HR 1 MIN	SBP 2 MIN	DBP 2 MIN	MAP 2 MIN	HR 2 MIN	SBP 3 MIN	DBP 3 MIN	MAP 3 MIN	HR 3 MIN	SBP 4 MIN	DBP 4 MIN	MAP 4 MIN	HR 4 MIN	SBP 5 MIN	DBP 5 MIN	MAP 5 MIN	HR 5 MIN
Cholesystectomy	Lap chole	51/M	51	M	80	2	Dexmeditomidin	150	82	105	60	158	100	119	100	144	92	109	92	136	89	105	81	132	85	101	75	124	80	95	71	116	72	87	61
Liver abcess	Liver abcess drainage	45/M	45	M	78	2	Dexmeditomidin	148	90	109	66	162	90	114	101	150	86	107	92	144	83	103	83	140	80	100	78	120	76	91	74	115	70	85	62
Fracture shaft of lt. ulna	ORIF lt. ulna	18/M	18	M	65	1	Dexmeditomidin	126	78	94	78	140	92	108	102	135	80	98	90	128	76	93	82	124	74	91	72	120	71	87	64	114	63	80	66
Cholesystectomy	Open chole	51/F	51	F	55	2	Dexmeditomidin	140	74	96	72	156	88	111	110	146	84	105	92	138	81	100	84	132	80	97	71	126	70	89	66	120	62	81	60
CSOM right ear	Tympanomastoidectomy	22/F	22	F	50	1	Dexmeditomidin	120	82	95	82	134	88	103	97	128	82	97	90	120	80	93	79	116	78	91	70	118	70	86	74	116	61	79	61
Cholesystectomy	Lap chole	34/M	34	M	70	1	Dexmeditomidin	130	78	95	86	138	90	106	111	130	82	98	97	124	78	93	88	120	75	90	80	119	68	85	80	114	60	78	80
Cholesystectomy	Lap chole	45/F	45	F	70	1	Dexmeditomidin	132	82	99	82	140	100	113	113	132	98	109	99	122	92	102	90	120	88	99	82	120	80	93	76	118	73	88	68
Umbilical hernia	Mesh Hernioplasty	48/M	48	M	72	2	Dexmeditomidin	152	84	107	52	160	90	113	121	156	86	109	102	142	85	104	91	135	83	100	83	122	72	89	79	117	65	82	70
DNS to rt.	Septoplasty	31/F	31	F	58	1	Dexmeditomidin	120	86	97	82	134	88	103	109	126	82	97	91	120	80	93	82	119	76	90	73	118	70	86	67	115	61	79	60
CSOM left ear	Tympanoplasty	33/M	33	M	78	1	Dexmeditomidin	122	80	94	74	130	86	101	107	128	82	97	99	122	81	95	90	118	79	92	80	117	88	98	76	116	76	89	70
Fracture maxilla + mandible	ORIF maxilla + mandible	29/M	29	M	80	1	Dexmeditomidin	130	82	98	72	140	82	101	106	136	80	99	96	130	79	96	90	125	75	92	82	124	86	99	75	117	80	92	66
Cholesystectomy	Lap chole	55/F	55	F	60	2	Dexmeditomidin	138	84	102	62	142	84	103	102	135	78	97	88	126	76	93	80	123	75	91	71	122	86	98	65	114	81	92	60
Cholesystectomy	Open chole	54/F	54	F	65	2	Dexmeditomidin	140	88	105	64	148	90	109	103	130	86	101	90	124	83	97	81	120	80	93	70	118	74	89	66	115	70	85	61
Cholesystectomy	Lap chole	45/M	45	M	68	2	Dexmeditomidin	142	82	102	51	170	110	130	110	160	100	120	91	142	96	111	83	132	92	105	72	124	81	95	62	119	76	90	56
Fracture head of humerus rt.	ORIF rt. Humerus	30/M	30	M	65	1	Dexmeditomidin	128	84	99	59	134	100	111	109	126	97	107	89	122	92	102	80	117	90	99	72	116	82	93	67	115	75	88	60
DNS to rt.	Septoplasty	25/M	25	M	68	1	Dexmeditomidin	124	86	99	82	130	88	102	99	120	84	96	80	118	83	95	78	116	81	93	70	116	72	87	65	114	68	83	61
Umbilical hernia	Hernioplasty	27/M	27	M	55	1	Dexmeditomidin	126	80	95	88	132	90	104	100	130	80	97	92	122	76	91	82	117	74	88	71	118	82	94	67	116	72	87	59
Cholesystectomy	Lap chole	30/F	30	F	50	1	Dexmeditomidin	122	78	93	76	136	94	108	112	128	84	99	98	121	81	94	88	119	79	92	80	120	70	87	74	116	68	84	68
Cholesystectomy	Lap chole	45/M	45	M	66	1	Dexmeditomidin	126	80	95	88	130	96	107	111	126	90	102	96	122	87	99	90	120	84	96	81	118	76	90	76	115	70	85	70
Cholesystectomy	Lap chole	56/F	56	F	50	2	Dexmeditomidin	138	82	101	89	146	102	117	113	140	100	113	99	132	87	102	91	123	85	98	83	122	75	91	78	119	69	86	69
Small bowel obstruction	Exploratory laprotomy	30/M	30	M	68	1	Dexmeditomidin	120	78	92	84	128	88	101	114	126	80	95	102	123	79	94	92	120	73	89	84	117	68	84	80	115	62	80	72
CSOM left ear	Tympanoplasty	28/F	28	F	60	1	Dexmeditomidin	110	74	86	86	126	90	102	120	120	86	97	101	121	83	96	94	117	80	92	85	116	72	87	79	114	67	83	71
Fracture head of humerus lt.	ORIF lt. humerus	22/F	22	F	62	1	Dexmeditomidin	114	76	89	74	130	88	102	122	124	82	96	98	122	80	94	91	121	76	91	80	119	71	87	72	114	66	82	64
Cholesystectomy	Lap chole	30/M	30	M	59	1	Dexmeditomidin	126	80	95	76	134	86	102	130	120	98	105	111	123	95	104	102	120	90	100	92	118	84	95	81	116	78	91	72
Cholesystectomy	Lap chole	33/F	33	F	50	1	Dexmeditomidin	130	80	97	82	140	78	99	121	130	80	97	115	124	79	94	103	121	75	90	91	120	70	87	72	117	60	79	63
Cholesystectomy	Lap chole	46/F	46	F	55	1	Dexmeditomidin	132	78	96	80	142	76	98	120	128	78	95	101	123	75	91	91	118	72	87	82	116	68	84	73	114	61	79	65
Umbilical hernia	Mesh Hernioplasty	45/M	45	M	70	1	Dexmeditomidin	128	78	95	84	136	84	101	118	126	78	94	99	120	76	91	90	116	74	88	83	119	66	84	75	116	60	79	62
DNS to rt.	Septoplasty	28/M	28	M	72	1	Dexmeditomidin	122	80	94	84	148	82	104	111	130	78	95	96	122	73	89	89	118	71	87	80	120	64	83	70	118	62	81	60
CSOM left ear	Tympanoplasty	34/F	34	F	50	1	Dexmeditomidin	130	82	98	86	156	90	112	112	140	80	100	98	134	74	94	91	128	72	91	85	124	65	85	71	120	61	81	64
Cholesystectomy	Lap chole	55/F	55	F	54	2	Dexmeditomidin	148	86	107	88	168	92	117	113	150	90	110	100	142	88	106	92	136	85	102	70	129	71	90	63	120	63	82	59
Cholesystectomy	Lap chole	58/M	58	M	68	2	Dexmeditomidin	144	88	107	78	160	88	112	100	154	80	105	82	140	76	97	78	131	75	94	70	125	64	84	62	119	60	80	57
Umbilical hernia	Mesh Hernioplasty	30/M	30	M	70	1	Dexmeditomidin	120	82	95	77	146	100	115	99	138	96	110	83	132	91	105	76	124	84	97	69	126	72	90	60	121	64	83	58
Cholesystectomy	Lap chole	48/M	48	M	72	2	Dexmeditomidin	152	90	111	74	170	98	122	109	158	90	113	89	145	87	106	81	134	83	100	73	128	73	91	68	121	66	84	60
Cholesystectomy	Lap chole	50/M	50	M	65	2	Dexmeditomidin	160	94	116	89	168	101	123	113	160	86	111	100	156	80	105	76	150	76	101	68	134	69	91	62	122	62	82	56